Echocardiographic Assessment of Left Ventricular Remodeling Process Among Acute Myocardial Infarction Patients

Mayyadah Dheyauldeen Mohsin*, Abbas Khalifa Ressan, Asaad Abdullah Abbas****

ABSTRACT:

BACKGROUND:

Echocardiography is use for assessment of left ventricular remodeling after myocardial infarction, which is the focus of this thesis. We investigated the influence of various traditional echocardiographic parameters to assess the process of left ventricular remodeling such as left ventricular volumes and ejection fraction, wall motion score index, mitral regurgitation and diastolic dysfunction

OBJECTIVE:

To invistigate the usefulness of two-dimensional echocardiography for accurate evaluation of left ventricular (LV) remodeling after acute ST elevation myocardial infarctions (STEMI).

METHODS AND RESULTS:

Two-dimensional echocardiography was performed within 5 days on a 100 patients admitted to the hospital with a first ST-elevation AMI. Several clinical and echocardiographic variables were analyzed. Baseline demographic data, blood pressure, and pulse were obtained. Various traditional echocardiographic parameters have been shown to provide diagnostic information, such as left ventricular volumes and ejection fraction, wall motion score index, mitral regurgitation and left atrial pressure. A left ventricular wall motion score index was derived from analysis of regional wall motion; an index of 1.5 or more within 5 days of admission identified patients at high risk for remodeling and LV dysfunction. Predictors of early LV remodeling were older age, male gender, history of diabetes mellitus or hypertension, high leukocyte count, high admission blood glucose level, high wall motion score and anterior location myocardial infarction.

CONCLUSION:

After acute ST-elevation myocardial infarction, early determination of the wall motion score index by two-dimensional echocardiography is useful for identifying patients at high risk for complications and to differentiate patients with and without development of LV remodeling accurately and early on the basis of wall motion score index as a measure of infarct size, a highly predictive variable.

KEYWORD : echocardiography, remodeling, myocardial infarction.

INTRODUCTION:

Acute myocardial infarction (MI) remains a leading cause of morbidity and mortality worldwide. A common clinical diagnostic classification scheme is based on electrocardiographic findings as a means of distinguishing between two types of MI, one that is marked by ST elevation (STEMI) and one that is not (NSTEMI). ST elevation myocardial infarctions (STEMI) constitute ~40% of all AMIs.[1]

Cardiac remodeling refers to the changes in size, shape, structure and physiology of the heart after injury to the myocardium.[2] The injury is typically due to acute myocardial infarction (usually transmural or ST segment elevation infarction).[3] Remodeling has been recognized by echocardiography as early regional infarct expansion (stretching, thinning and bulging).[4]The noninfarcted region also undergoes an important lengthening that may cause elevation of diastolic and systolic wall stresses.[5] As the geometry of the left ventricle changes, it becomes less elliptical and more spherical. There are also changes in ventricular mass and volume, both of which may adversely affect cardiac function.[6] The extent of ventricular enlargement after infarction is related to the magnitude of the initial damage to the myocardium,7 and, if not attenuated or reversed by intervention, has a poor prognosis.[7]

*Baghdad Teaching Hospital.
**Ibn –Alnafees Hospital

AIM OF THE STUDY:

The purpose of this present study was to characterize and evaluate the prevalence and pattern of left ventricular (LV) remodeling after acute transmural myocardial infarction (STEMI) including changes in LV volumes, ejection fraction (EF), regional wall motion score index (RWMSI) and left ventricular diastolic function, and to identify clinical and laboratory biomarkers, and evaluate echocardiographic criteria that may predict development of acute LV remodeling.

PATIENTS AND METHODS:

This is a cross sectional study done in Al-Shaheed Al-Sadr general hospital from April 2014 to May 2015. In total, hundred (100) patients who were admitted to the Al-Shaheed Alsadr general hospital with STEMIs were included in this study. All participants were examined in the Coronary Care Unit by echocardiography within 5 days after STEMI.

Myocardial infarction was diagnosed according to Third Universal Definition of Myocardial Infarction by ESC/ACCF/AHA/WHF Expert Consensus Document on 2012:

The detection of a rise and/or fall of cardiac biomarkers, with at least one of the values being elevated (>99th percentile upper reference limit, or URL). The highly sensitive and specific cardiac troponin (cTn) is the approved biomarker of myocardial necrosis. In addition, one of the five following predefined criteria : (1) symptoms of myocardial ischemia; (2) new (or presumably new) significant ST-segment/T-wave changes or left bundle branch block; (3) development of pathological Q waves on ECG; (4) new loss of viable myocardium or regional wall motion abnormality by imaging; (5) identification of intracoronary thrombus by angiography or autopsy.8

, I had performed Echocardiography using Vivid 7 ultrasound systems (GE Ultrasound) with a 3.5-MHz transducer. All participants were examined using conventional two-dimensional echocardiography and Pulse wave DTI according to standardized protocols. All echocardiograms were stored digitally and analyzed offline.

Statistical analysis

The statistical analysis of the quantitative variables has been carried out through the description of the minimum and maximum values, and of the calculation of the standard deviations and averages. In relation to the qualitative variables, the absolute and relative frequencies have been calculated. The analysis of correlation between various findings and the two-dimensional echocardiographic findings has been carried out by using the Pearson's correlation method (r), 95% of confidence interval. A two-tailed p-value <0.05 or <0.01 were considered statistically significant. The data were analyzed using IBM SPSS statistics version 22 for Windows

RESULTS:

Hundred (100) patients who were admitted to the hospital with STEMIs were examined by echocardiography within 5 days.

Clinical evaluation; We determined the location of the myocardial infarction from the standard electrocardiographic criteria was anterior in 62 patients, posterior in 38 patients (21 inferior, 2 inferoposterior and 15 infero-postero-lateral)

In the present study we determined the final size of the myocardial infarction by calculation of the wall motion score index (WMSI). A wide range of wall motion abnormalities was noted, ranging from hypokinesia to dyskinesia. The wall motion score index (WMSI) ranged from 0.11 to 2.2 (mean 1.2 ± 0.5) .

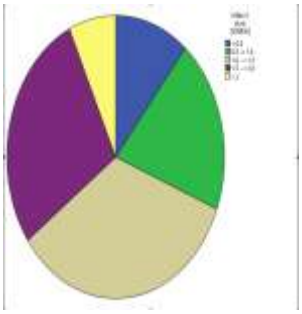

Figure 4: The Distribution of WMSI in the Study group.

There was a positive correlation between the wall motion score index with age and a significant correlation (P <0.05) with gender

Patients with larger infarct size and the consequent remodeling are mostly likely male and older in age than female gender or younger age group who are more likely to have a smaller infarct size.

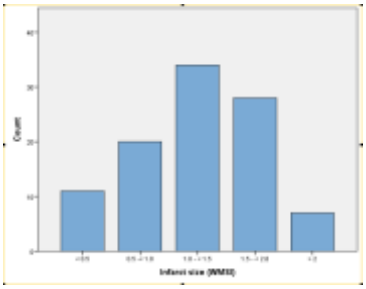

Figure 6: Infarct Size by the WMSI.

Correlation of initial wall motion score index with clinical variables.

There was a positive correlation between the wall motion score index and smoking and the body weight status. The wall motion score index was significantly higher in the diabetics (P <0.01),almost 69% of the remodeling group were diabetics vs 31 % in the patients who were not diabetic (figure 8), also it was significantly higher in the hypertensives (P < 0.01) with 63% of the remodeling group being hypertensive, (figure 9), or both. There was a positive relation between the remodeling process and history of smoking as 60% of the remodeling group were current smokers (figure 10) and all of them were overweight (BMI ≥ 25)

of interest, less than one third of the patients with AMI in the study group had been treated with the thrombolytic therapy, with approximately 25 % in the remodeling group in contrast to 35% of patients who had no remodeling (figure 12), this management strategy may have a role in the development of remodeling and thus LV dysfunction after AMI.

Correlation of initial wall motion score index with cardiac markers and the laboratory variables.

There was a positive correlation between the wall motion score index and the cardiac markers. Troponin level (P <0.01), Myoglobin (P <0.01) and Creatine kinase (P <0.01) was significantly more positive in the remodeling group of patients with higher wall motion score index. There was a significant correlation between the wall motion score index and fasting blood sugar (P<0.05) with a linear relationship between the degree of abnormal glucose metabolism and each marker of LV dysfunction (LV end-diastolic volume, LV end-systolic volume, LV ejection fraction, and diastolic mitral annular velocity ratio [E/e']), the same is

applicable to the leukocyte count, (P<0.01) with the higher WMSI infarct having a higher count of leukocyte, on the other hand patients with a lower level of hematocrit had a greater likelihood of a larger infarct size (P<0.05). There was positive but non-significant relation of infarct size with other laboratory variables as Serum creatinine, ALT (SGPT), AST (SGOT), Total serum cholesterol and theTriglyceride level.

Echocardiography and Infarct Size

A significant correlation was found between all parameters of LV systolic function and final infarct size. Importantly, the echocardiographic parameters of LV systolic function were able to discriminate between patients with remodeling and non-remodeling. The remodeling group have significantly higher Infarct size (WMSI) and positively related to LV-EDVI (P <0.01) and LV-ESVI (P < 0.01) and inversely related to LV-EF (PV < 0.01).

The wall motion score index was significantly higher in patients with anterior myocardial infarction(1.4 ± 0.4) than in those with inferior infarction (0.9 ± 0.6) with the mean of 1.2 ± 0.5, (P <0.0), figure 13. In other words infarct location can predict infarct size or WMSI. 80% of the remodeling group had anterior MI, in contrast to 20% of the posterior MI patients with large infarct.

Eighty % of the patients in the remodeling group had moderate or severe diastolic dysfunction with significant correlation with the infarct size (PV <0.01), also there was a significant correlation between E/e′ ratio and infarct size (PV <0.05), the same apply for the LAVI with a PV <0.01,

Mild to severe mitral regurgitation was present in 66% of the remodeling group compared with 34% of the patients with smaller infarcts size, it correlated significantly with the degree of WMSI and consequently the infarct size (PV <0.01),

Eleven (11) patients (19%) with posterior infarction were accompanied with right ventricular infarct manifested as RV hypokinesia in contrast with only 4 patients with anterior infarct having RV hypokinesia.

Table 1: Markers by Infarct Size (WMSI).

Characteristics	Patients with Remodeling WMSI ≥ 1.5 (n = 35)	Patients with no Remodeling WMSI < 1.5 (n =65)	Correlation
Troponin	34 (97%)	52 (80%)	Sig. P <0.01
Creatine kinase	28 (80%)	44 (68%)	Sig. P <0.01
Myoglobin	35 (100%)	59 (90%)	Sig. P<0.01
Leukocyte count (N ≤ 10.0 X 10^9/L)	15.0 X 10^9	11.2 X 10^9	Sig. P <0.01
Hematocrit %	38	43	Sig. P< 0.05
Fasting blood sugar (N ≤ 5.6 mmol/l)	13.2	8.5	Sig. P< 0.05
Serum creatinine (N < 120)	78	73	NS
ALT (SGPT) (N= ≤ 36 U/L)	41	39	NS
AST (SGOT) (N ≤ 35 U/L)	85	71	NS
Total serum cholesterol (N ≤ 5 mmol/l)	4.5	4.4	NS
Triglyceride (N < 1.7 mmol/l)	1.5	1.4	NS

Table 2:Markers by Infarct Size (WMSI).

Characteristics	Patients with Remodeling WMSI ≥ 1.5 (n = 35)	Patients with no Remodeling WMSI < 1.5 (n =65)	Correlation
Troponin	34 (97%)	52 (80%)	Sig. P <0.01
Creatine kinase	28 (80%)	44 (68%)	Sig. P <0.01
Myoglobin	35 (100%)	59 (90%)	Sig. P<0.01
Leukocyte count (N ≤ 10.0 X 10^9/L)	15.0 X 10^9	11.2 X 10^9	Sig. P <0.01
Hematocrit %	38	43	Sig. P< 0.05
Fasting blood sugar (N ≤ 5.6 mmol/l)	13.2	8.5	Sig. P< 0.05
Serum creatinine (N < 120)	78	73	NS
ALT (SGPT) (N= ≤ 36 U/L)	41	39	NS
AST (SGOT) (N ≤ 35 U/L)	85	71	NS
Total serum cholesterol (N ≤ 5 mmol/l)	4.5	4.4	NS
Triglyceride (N < 1.7 mmol/l)	1.5	1.4	NS

Echocardiographic Findings by Infarct Size (WMSI)			
Characteristics	Patients with Remodeling WMSI ≥ 1.5 (n = 35)	Patients with no Remodeling WMSI < 1.5 (n =65)	P Value
LVEF %	33 ± 8	49 ± 8	Sig. P < 0.01
LV EDV (mL/m^2)	75 ± 18	56 ± 13	Sig. P < 0.01
LV ESV (mL/m^2)	51 ± 14	29 ± 9	Sig. P < 0.01
E/e′ ratio	15.1 ± 5.1	12.8 ± 5.6	Sig. P < 0.05
LAP	21	18	Sig. P < 0.05
Moderate or severe DD	28 (80%)	35 (53%)	Sig. P < 0.01
Left atrial volume index (mL/m^2)	34 ± 5	22 ± 6	
Mild - severe MR	23 (66%)	22 (34%)	Sig. P < 0.05
Mild TR with PH	7 (21%)	12 (17%)	NS
RV hypokinesia	7 (20%)	7 (10%)	

Table 3: Echocardiographic Findings by Infarct Size (WMSI).

Characteristics	Patients with Remodeling WMSI ≥ 1.5 (n = 35)	Patients with no Remodeling WMSI < 1.5 (n =65)	P Value
LVEF %	33 ± 8	49 ± 8	Sig. P < 0.01
LV EDV (mL/m^2)	75 ± 18	56 ± 13	Sig. P < 0.01
LV ESV (mL/m^2)	51 ± 14	29 ± 9	Sig. P < 0.01
E/e′ ratio	15.1 ± 5.1	12.8 ± 5.6	Sig. P < 0.05
LAP	21	18	Sig. P < 0.05
Moderate or severe DD	28 (80%)	35 (53%)	Sig. P < 0.01
Left atrial volume index (mL/m^2)	34 ± 5	22 ± 6	
Mild - severe MR	23 (66%)	22 (34%)	Sig. P < 0.05
Mild TR with PH	7 (21%)	12 (17%)	NS
RV hypokinesia	7 (20%)	7 (10%)	

DISCUSSION:

The left ventricular remodeling after AMI reflects the mechanical modifications of adaptation of the LV in the face of the ischemic event, being a factor of important prognosis in the clinical evolution of the patients.[9] The left ventricular dilatation, as well as the reduction in the ejection fraction of the LV represent important factors of poor prognosis related to the heart failure after AMI.

Our study confirmed the use of two dimensional echocardiography in predicting remodeling in

STEMI patients. Multiple studies have established its use in predicting regional [10] as well as global LV remodeling early after MI. Many studies have confirmed the rapid deterioration of LVF after acute coronary occlusion.[11]This has important clinical implications because it may provide a means for early identification of patients who have substantial infarction.

Infarct Size and LV remodeling

The size of the myocardial infarction as determined by the wall motion score index (WMSI) is an established parameter of LV systolic function, validated as a prognostic indicator after MI.[12]

The WMSI has been previously validated in several echocardiographic studies and correlated well with LVEF.[13,14,15] It is a strong predictor of mortality and major adverse cardiovascular events,[16] and has been found to be one of the most important determinants of infarct expansion and ventricular remodeling.[17]

The principal finding of this study, according to the all echocardiographic parameters of left ventricular function, was that a threshold level of infarct size (WMSI ≥1.5) almost always resulted in LVR.

We found a significant correlation between echocardiographic parameters of LV systolic function and the remodeling process of STEMI. We have shown that WMSI determinants of infarct size measured early after MI predict LVR

In concordance with other studies, a significantly larger infarct size, was observed in patients with subsequent remodeling, EEK C et al found wall motion score index >1.3 accurately identified patients with substantial infarct.[18] An another study Tarantini G, et al demonstrated that below a threshold of 1.5 of wall motion score index apparently does not affect the LVEF and therefore a safety threshold could be set at this value.[19]

In the present study, 35% of the patients have presented echocardiographic evidence of ventricular remodeling within the hospitalization period. End-diastolic volume indexed to body surface area (EDVi) (P<0.01) and end-systolic volume (ESVi) (PV< 0.01) were significantly increased compared to patients without remodeling (n=35), while it was inversely related to LV-EF (PV< 0.01). LVESVI was the most predictive parameter indicating LV remodeling after AMI.In the remodeling group there was also a strong, nearly significant, tendency for more anterior AMIs.

Relation between WMSI, LVEF, ESV and EDV

Infarct size measured with WMSI was correlated with LVEDV and LVESV and, as other studies stated, was a major predictors of LV function, geometry, and outcome after MI.[12,20]. LV systolic function appears to depend on the degree and severity of regional LV function.

Multiple studies as Gaudron P, et al and Korup E, et al had found a strong relation between remodeling and LVEF, EDVI, ESVI.[5 ,21] MannL. indicated that LV volumes predict outcome more reliably than does the EF.[22] The incidence of remodeling was closely linked to the rise in LVESVI and LVEDVI beyond 51 mL/m^2 and 75 mL/m^2 respectively. Patients with LVR had significantly lower LVEFs (33% vs. 49%, P < 0.01), and higher WMSI (mean 1.78 vs 1.23, P < 0.01).

Relation between anterior and non-anterior MI

This study, as similar other studies (Moller JE, et al. Orn S, et al) [16,47]demonstrated that patients with anterior MI have worse post-infarction LV remodeling with larger infarct size than inferior infarcts (P < 0.01), worse regional and global LV systolic function than those with non-anterior MI (P <0.01).

Myocardial infarctions involving higher number of left ventricular wall segments most commonly occurred in the anterior MI, however, we found that AMI involving the inferior, posterior and the lateral walls was also associated with a larger infarct size. In another study, Bolognese L et al showed that the larger myocardial necrosis in anterior than in non-anterior MI patients, suggesting that MI size but not its location was an independent predictor of post-infarction remodeling.[24] Masci P G suggested that MI location was not accounted for MI size and thus the independent contribution made by MI location likely reflected the larger amount of myocardial necrosis intrinsic to anterior infarcts.[49]

Diastolic dysfunction and infarct size in AMI

Pulmonary congestion after infarction reflects raised LV filling pressures and may involve impaired active relaxation of the myocardium and increased LV chamber stiffness and hence abnormalities in diastolic function.[26]

E/e' appeared to be a particularly useful indicator of elevated LV filling pressure, [25] In a retrospective study of 250 patients with AMI; Hillis GS et al showedan increased E/e′ ratio >15 was an important predictor of LVR and incremental to LVEF, age, and a restrictive transmitral filling pattern.[27] Prior data (Alam M, et al) suggested that E/e' ratio is the least affected by the location of infarction.[28]

Studies have investigated the relation between LA dilatation and the remodeling after AMI, but didn't find a relation.[29]

Mitral regurgitation and the infarct size in acute MI

Ischemic mitral regurgitation (IMR) occurs after coronary artery disease (CAD) in the absence of intrinsic valve lesions and is frequent after MI.[30,31]Lehmann et al. studied 206 patients within 7 hours after first myocardial infarction using contrast left ventriculography and demonstrated the presence of mitral regurgitation in 13% of patients.[32]

There was a significant correlation between occurrence of mitral regurgitation and LV remodeling as almost two thirds of the patient with LV remodeling had MR of varying degree in contrast to less than one third of the patients with no LV remodeling. Many echocardiographic studies of the patients with IMR as (AmigoniM,et al**)** indicated that those with MR had a lower EF, larger left ventricular volumes and greater wall-motion score index, in comparison with patients without MR,[33] exactly as we found in our study.

Emerging concepts on the pathophysiology of ischemic MR have shown that it may be more related to local and global ventricular remodeling with worse LV function, greater LV enlargement and chamber distortion.[34]

However, in our study there were 6 patients with inferior or infero-posterior myocardial infarcts associated with moderate to severe MR who have a normal LV volumes and EF and small infarct size. In consistent with our findings recent studies have raised the question of whether global LV remodeling represents the main determinant of post-MI MR, because similar degree of LV dysfunction or enlargement might be associated with widely different degree of MR.[33] Indeed, localized remodeling of the posterior walls leads to papillary muscle displacement and results in increased mitral tenting independent of LV volumes.[34] Moreover, varying degrees of leaflet adaptation to LV remodeling may play a role in the development of functional MR.[33]

In our study Troponin, myoglobin and CK were strongly correlated with the infarct size as many studies have suggested. We investigated whether a high white blood cell (WBC) count in acute myocardial infarction (AMI) may be associated with a higher risk of left ventricular (LV) remodeling; WBC counts were significantly higher in patients who underwent LV remodeling. A high WBC count on admission for AMI is an independent predictor of LV remodeling.[34]

A significantly higher proportion of diabetic or hypertensive subjects developed left ventricular remodeling. Patients with diabetes mellitus, hypertension or history of smoking had markedly higher wall motion score index (WMSI) ($P < 0.01$) with positive relation with left ventricular systolic and diastolic function as multiple studies (Celik T, et al and Hajsadeghi S, et al) have suggested.[36 ,37]

Obesity and the infarct size in acute MI

There was a positive but non-significant relation between the body mass index and the infarct size, with a tendency to a larger LV-ESVI in the obese patients, but not to other parameters of LV systolic and diastolic function. Studies have suggested as (Poyraz F, et al) that no unfavorable effects of overweight on the left ventricular systolic and diastolic function.[38]

CONCLUSION:

The results of this study prove the incremental prognostic benefits of echocardiographic imaging after acute MI

Various parameters derived from 2D echo had demonstrated to provide information on the possible occurrence of LV remodeling, including LV volumes, LVEF, WMSI, and E/e' ratio.

These parameters focused on LV function, but additional information on cardiac performance such as MR, and RV function were also important for evaluation of LVF

Early prediction of LVR may be helpful in identifying patients at higher risk for arrhythmias, congestive heart failure, who would benefit most from careful follow-up and aggressive treatment strategy.

In addition, the use of thrombolytic therapy in the setting of acute MI proved to be very efficient way in the prevention of development of LV remodeling, which should intensify the written guideline for its use.

REFERENCES:

1. Sutton, MG and Sharpe, N. Left ventricular remodeling after myocardial infarction: pathophysiology and therapy. Circulation. 2000;101:2981–88.
2. Tarantini G, Razzolini R, Cacciavillani L, et al. Influence of transmurality, infarct size, and severe microvascular obstruction on left ventricular remodeling and function after primary coronary angioplasty. Am J Cardiol. 2006;98:1033-40.

3. Pirolo JS, Hutchins GM, Moore GW. Infarct expansion: pathologic analysis of 204 patients with a single myocardial infarct. J Am Coll Cardiol1986;7:349–54.
4. Mollema,S.A, Nucifora G. Prognostic value of echocardiography after acute myocardial infarction. Heart 2009;95:1732-45.
5. Korup E, Dalsgaard D, et al. Comparisons of degrees of left ventricular dilation within 3 hours and up to 6 days after onset of first acute myocardial infarction. Am J Cardiol 1997;80:449.
6. Cohn J, Ferrari R, et al. Cardiac Remodeling—Concepts and Clinical Implications: A Consensus Paper From an International Forum on Cardiac Remodeling. J Am CollCardiol 2000;35:569–82.
7. Moller JE, Hillis GS, Oh JK, Reeder GS,et al. Wall motion score index and ejection fraction for risk stratification after acute myocardial infarction. Am Heart J 2006;151:419-25.
8. Feinberg MS, Schwammenthal E, Shlizerman L et al. Prognostic significance of mild mitral regurgitation by color Doppler echocardiography in acute myocardial infarction. Am J Cardiol 2000;86:903-7.
9. Lamas GA, Mitchell GF, Flaker GC, et al. Clinical significance of mitral regurgitation after acute myocardial infarction. Circulation.1997;96:827–33.
10. Main, M.L., Magalski, A., Chee, N.K., et al. Full-motion pulse inversion power Doppler contrast echocardiography differentiates stunning from necrosis and predicts recovery of left ventricular function after acute myocardial infarction. J Am CollCardiol. 2001;38:1390–94.
11. Main M.L., Hannen, M.N.,et al. Myocardial Contrast Echocardiographic Estimates of Infarct Size Predict Likelihood of Left Ventricular Remodeling After Acute Anterior Wall Myocardial Infarction. 2006;19:1.64-70.
12. Lebeau R., Serri K., Morice M, et al. Assessment of left ventricular ejection fraction using the wall motion score index in cardiac magnetic resonance imaging. 2012;105:91–98.
13. J.H. McGowan, J.G. Cleland. Reliability of reporting left ventricular systolic function by echocardiography: a systematic review of 3 methods. Am Heart J, 2003;146:388–97.
14. R.D. Rifkin, H. Koito. Comparison with radionuclide angiography of two new geometric and four nongeometric models for echocardiographic estimation of left ventricular ejection fraction using segmental wall motion scoring. Am J Cardiol;1990:1485–90.
15. R. Lebeau, M. Di Lorenzo, R. Amyot, et al. A new tool for estimating left ventricular ejection fraction derived from wall motion score index Can J Cardiol, 2003;19:397–404.
16. Stanton T, Leano R, Marwick T. Prediction of all-cause mortality from global longitudinal speckle strain: comparison with ejection fraction and wall motion scoring. CircCardiovasc Imaging. 2009;2:356–64.
17. Modena MG, Rossi R, Sgura FA, et al. Early predictors of late dilatation and remodeling after thrombolyzed anterior transmural infarction. Clin Cardiol1997;20:28–34.
18. Eek C, Grenne B,Brunv H, MD, et al. Strain Echocardiography and Wall Motion Score Index Predicts Final Infarct Size in Patients With Non–ST-Segment–Elevation Myocardial Infarction. Circulation: Cardiovascular Imaging. 2010;3:187-94.
19. Tarantini G, Razzolini R, Cacciavillani L, et al. Infarct size, and severe microvascular obstruction on left ventricular remodeling and function after primary coronary angioplasty. Am J Cardiol. 2006 15;98:1033.
20. Staat P, Rioufol G, Piot Cet al. Postconditioning the human heart. Circulation.2005;112:2143–48.
21. Gaudron P, Eilles C, Kugler I, Ertl G. Progressive left ventricular dysfunction and remodeling after myocardial infarction. Circulation 1993;87:755– 63.
22. MannL, Bogaev R, Buckberg D. Cardiac Remodelling and Myocardial Recovery: Lost in Translation? Eur J Heart Fail. 2010;12:789-96.
23. Orn S, Manhenke C, Anand IS, et al. Effect of left ventricular scar size, location, and transmurality on left ventricular remodeling with healed myocardial infarction. Am J Cardiol 2007;99:1109–14.
24. Bolognese L et al. Left Ventricular Remodeling After Primary Coronary Angioplasty Patterns of Left Ventricular Dilation and Long-Term Prognostic Implications. Circulation. 2002;106: 2351-57.

25. Masci P G, Ganame J, Francone M, et al. Relationship between location and size of myocardial infarction and their reciprocal influences on post-infarction left ventricular remodeling. European Heart Journal 2011;32:1640–48.

26. Whalley GA, Gamble GD, Doughty RN. Restrictive diastolic filling predicts death after acute myocardial infarction: systematic review and meta-analysis of prospective studies. Heart 2006;92:1588-94.

27. Hillis GS, Moller JE, Pellikka PA, et al. Noninvasive estimation of left ventricular filling pressure by E/e′ is a powerful predictor of survival after acute myocardial infarction. J Am CollCardiol. 2004;43:360–67.

28. Alam M, Wardell J, Andersson E, et al. Effects of first myocardial infarction on left ventricular systolic and diastolic function with the use of mitral annular velocity determined by pulsed wave Doppler tissue imaging. J Am SocEchocardiogr. 2000;13:343–52.

29. Dokainish H, Zoghbi A, Lakkis M, et al. Optimal noninvasive assessment of left ventricular filling pressures (a comparison of tissue Doppler echocardiography and B-type natriuretic peptide in patients with pulmonary artery catheters). Circulation. 2004;109:2432–39.

30. Yiu S, Enriquez-Sarano M, Tribouilloy C, et al. Determinants of the degree of functional mitral regurgitation in patients with systolic left ventricular dysfunction: a quantitative clinical study. Circulation 2000;102:1400–6.

31. Grigioni F, Enriquez-Sarano M, et al. Ischemic mitral regurgitation: long-term outcome and prognostic implications with quantitative Doppler assessment. Circulation 2001;103:1759–64.

32. Lehmann KG, Francis CK, Dodge HT. Mitral regurgitation in early myocardial infarction. Incidence, clinical detection, and prognostic implications. TIMI Study Group. Ann Intern Med 1992;117:10-7.

33. Amigoni M, Meris A, Thune J, et al. Mitral regurgitation in myocardial infarction complicated by heart failure, left ventricular dysfunction, or both: prognostic significance and relation to ventricular size and function. Eur Heart J. 2007;28:326–33.

34. Bursi F, Enriquez-Sarano M, et al. Heart Failure and Death after Myocardial Infarction in the Community. Circulation. 2005;111 295-301.

35. Bauters A, Ennezat V, et al ,Relation of Admission White Blood Cell Count to Left Ventricular Remodeling After Anterior Wall Acute Myocardial Infarction. J Am SocEchocardiogrphy2007;100:182–84.

36. Celik T, Turhan H, Kursaklioglu H. Impact of metabolic syndrome on myocardial perfusion grade after primary percutaneous coronary intervention in patients with acute ST elevation myocardial infarction. Coron Artery Dis. 2006 May;17:339-43.

37. Hajsadeghi S, Chitsazan M, Chitsazan M et al. Metabolic Syndrome is Associated With Higher Wall Motion Score and Larger Infarct Size After Acute Myocardial Infarction. Res Cardiovasc Med. 2015 Feb 20;4:2501-8.

38. Poyraz F, Kocaman S, et al Effect of overweight and obesity on the left ventricular systolic and diastolic functions in patients with acute myocardial infarction. Clin Intern Med 2012;35:229-36.